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APPENDIX 5

THE ROYAL SOCIETY
OF CANADA

LEAD IN GASOLINE

ALTERNATIVES TO LEAD IN GASOLINE

S U P P L E M E N T A R Y   R E P O R T

(1) COMMISSION'S CONCLUSIONS

(2) TECHNICAL APPRAISAL BY

Marcus C.B. Hotz
(Chief Scientist)

THE COMMISSION ON LEAD IN THE ENVIRONMENT

FEBRUARY 1986

CONTENTS

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Technical Appraisal

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TERMS OF REFERENCE

COMMISSION ON LEAD IN THE ENVIRONMENT

Purpose

To provide the Minister of the Environment with independent advice on the present and future risks and areas of concern resulting from the presence of lead in the Canadian environment, and

To consider and propose, as may be required, corrective measures to reduce the environmental and health risks associated with lead which may be adopted either independently by the Minister of the Environment or in cooperation with others. The effects of lead in the workplace are specifically excluded from these terms of reference.

Considerations

To fulfil these objectives the Commission is to report on the following:

1. (a) the sources of lead releases in Canada and their relative contributions;

   (b) the pathways by which lead enters the Canadian environment and the means and media by which lead is transported within the environment and to humans;

   (c) the toxicity of lead;

   (d) the potential or actual exposure of, and risks to human and environmental targets in Canada;

2. (a) practical corrective measures, as may be required, to reduce the risks associated with lead based on the Commission's review and assessment of the impact and implications of lead for the environment and for humans;

   (b) the economic, technical, social (where appropriate) and labour implications of reductions in lead releases and exposure from all sources including the implications of eliminating lead in gasoline.

Duration and Reporting

The Commission will sit for three years and provide a progress report by September 1, 1985 on the direction and findings of its review, and a final report including its recommendations by September 1, 1986. The Commission shall continue to sit for a third year to receive and review comments on its reports and to make recommendations on those comments.

The Commission's progress and final reports will be made available to the public.

All written submissions made to the Commission except those deemed by the submittor to contain confidential business information will be made available to the public.

PREFACE

In September, 1985, the Commission reported to the Minister of the Environment on lead in gasoline. Recommendation 12 of that report said that the environmental and health implications of the different octane sources (other than lead) needed to be explored further. This supplementary report presents such a study, and also looks at alternative fuels that may partially replace gasoline in Canadian use.

The Commission decided to proceed as follows:

(i) to ask the Chief Scientist, Dr. M.C.B. Hotz, to prepare a technical appraisal of the actual and potential problems; and

(ii) to present its own conclusions, based largely on Dr. Hotz' overview, on the use of these additives and alternatives in producing unleaded gasoline of suitable octane rating.

These materials are presented in reverse order, so that the reader can get a quick overview of the Commission's opinions. Among these is that little attention has been given to the question. A large volume of reports deals with the health hazards of leaded gasoline. Much less is known about the consequences of <u>removing</u> lead.

The Commission appreciates the help received from National Health and Welfare Canada, the United States Environmental Protection Agency (US EPA) and the Petroleum Association for Conservation of the Canadian Environment (PACE).

F. Kenneth Hare
CHAIRMAN

THE COMMISSION'S CONCLUSIONS

1. The Commission finds that there are many potential alternatives to tetraethyl lead as octane enhancers. Some, however, may have adverse health effects. The pace of conversion should be deliberate enough to allow choice of methods that will ensure that the penalties paid do not exceed the expected health gains, by releasing other toxic substances into the environment. [see Technical Appraisal (TA) pp.1-5]

2. For example, as tetraethyl lead is replaced as an octane source in gasoline by more severe reforming and isomerization, higher concentrations of aromatic hydrocarbons will arise. Such compounds will be present whatever other additives are used, and may themselves be used as additives. Most of the automotive emissions of these compounds will be toluene and xylenes, which are rapidly metabolized and excreted from the human body. Benzene will also be present, however. This aromatic is a potent carcinogen as well as an excellent octane enhancer. [(TA), pp. 11-14].

3. Market trends brought about by the rapid phasedown of lead in the United States have already made the aromatics more valuable as octane enhancers. We are concerned that the proportion of benzene in gasoline, which is unregulated in both Canada and the United States, will consequently increase. The Commission recommends, therefore, that the Government of Canada evaluate the health effects of increased benzene in motor gasoline, and consider establishing limits on the allowable concentration. [TA, pp. 13-18]

4. <u>Methyl cyclopentadienyl manganese tricarbonyl</u> (MMT) is currently allowed as an octane enhancer in all gasolines in Canada, but is prohibited in unleaded fuel in the United States. After considering the evidence summarized in the following technical appraisal, <u>we find that the current-technology catalysts are unlikely to be damaged or rendered inoperative by the use of this compound at the present federal standard concentration (.018 grams of manganese per litre).</u> [TA, p. 7]

5. MMT also has implications for human health. Although highly toxic in its pure form, it does not normally present an occupational handling problem at the very low concentrations used in gasoline. It is almost completely combusted to manganese oxides in the automobile engine, so that the additive itself is not a hazard. The toxicity of manganese to the central nervous system and other organs at very high levels of exposure is well known. So are the effects of manganese deficiency, at the other end of the spectrum. [TA, pp. 6-8].

6. Manganese is one of the more abundant elements in the earth's crust, and its high concentration in soils, water and air reflects this fact. National Health and Welfare Canada has predicted that <u>the average additional individual intake of manganese resulting from the use of MMT in gasoline is likely to be no greater than 0.3 micrograms per day (µg/day). This compares with an average individual uptake from food, water and respiration of about 100-140 µg/day. By comparison with these large amounts already handled by the body, the extra loading on the public at large from MMT is and will remain very small.</u> [TA, pp. 10-11].

7. Methyl tertiary butyl ether (MTBE) is increasingly being used as an octane enhancer in the United States. It is totally miscible with gasoline so that, unlike methanol (from which it is made), it does not require a cosolvent (such as ethanol or tertiary butanol) to prevent it from dissolving in any moisture that may be present in the fuel system and separating out into two liquid phases. The oxidation products of blends of MTBE and gasoline are broadly similar to those of gasoline. [TA, pp. 22-23]

8. The Commission hence concludes that MTBE is safe to use and is an attractive option, though its manufacture implies an energy penalty by comparison with methanol itself. Isobutylene, the other feedstock used in its manufacture, is in short supply in Canada, so that MTBE is likely to be imported, with a negative impact on the balance of payments. Health and environmental problems associated with its use in automotive fuels do not appear to be any greater than those for gasoline. [TA, pp. 22-23]

9. Canada is one of the world's largest producers of methanol, which is the only alcohol additive or alternative fuel that is a viable option for this country. It can be used either as a blend with gasoline, usually at concentrations of 5%, or as an alternative fuel (85-100%). Methanol is itself a toxin, whose oxidation leads to increased emissions of formaldehyde, a toxic compound that is also reported to have carcinogenic properties at high exposure levels. Formaldehyde and methanol emissions can be easily oxidized by catalytic converters, however, and we believe that they do not represent a health hazard at

the low concentrations found in the emissions from methanol or methanol blends. [TA, pp. 23-26]

10. The use of neat methanol fuels involves technologies that can only be factory-fitted at the time of manufacture. Otherwise, corrosion and damage to the fuel system will ensue. The emission catalyst system of such vehicles will be tailored to control the formaldehyde formed. Methanol/gasoline blends, on the other hand, are suited to the existing vehicle fleets. [TA, pp. 28-33]

11. Propane has become fairly popular as an alternative fuel. Although it has been widely used for fleets of delivery vehicles and taxis, we doubt whether it is likely to become a significant alternative to gasoline. Propane-fuelled vehicles appear to be economical to run, but maintenance of these retrofitted gasoline vehicles will probably be costly in the long run, as their fuel systems were never designed to run on propane. Regulated emissions (CO, NO_x, hydrocarbons) are generally lower than for gasoline engines, but there is little information on the health effects of propane, other than that it is an asphyxiant in high concentrations. It is probably safe to assume that emissions are likely to comprise a quite small number of simple compounds, given that the propane molecule has only three carbon atoms, and that the fuel itself is fairly pure and not a mixture of hundreds of compounds as is gasoline. [TA, pp. 33-35]

12. Compressed natural gas is mainly methane. Its oxidation products are predominantly CO, CO_2 and water, with some NO_x and possibly formaldehyde, depending on the fuel-air mixture. Among its main advantages are the direct use of natural gas and the fact that, in

principle, vehicles can be refueled overnight through the domestic and industrial gas distribution system. The radical difference from other fuels makes retrofitting impossible so that the system has to be factory designed and built. We believe that these advantages are outweighed by the need for vehicles to carry high pressure tanks, and the cost and possible danger of operating high-pressure home pumping stations. (TA, pp. 35-36)

T E C H N I C A L   A P P R A I S A L

by

Marcus C.B. Hotz

TECHNICAL APPRAISAL

By Marcus C.B. Hotz

Introduction

In its interim report, Lead in Gasoline: a Review of the Canadian Policy Issue, the Commission reviewed the health concerns and economic effects that arise from the presence of lead in gasoline, and its phasedown (RSC, 1985). During its investigations, the Commission accepted the view that to reduce or eliminate lead from gasoline by switching to lower octane fuels, and to engines with lower compression ratios, was unrealistic. To do so would be both uneconomic and inefficient in terms of fuel consumption. If tetraethyl lead is no longer to be used as an octane enhancer in gasoline, or its role restricted, other octane sources have to be found. The health and environmental impacts of such substitutions have to be evaluated. It would be unwise to substitute another set of problems for those posed by lead. This question is thus within the Commission's terms of reference, even though it does not arise from lead directly.

Fuel octane can be matched to engine compression ratios in several ways or combinations of ways:

(i) more severe refining of petroleum fuels, converting low octane hydrocarbon molecules into differently structured but chemically similar molecules with higher octane values by reforming, cracking, alkylation and isomerization (PACE, 1985);

(ii) replacing tetraethyl lead in the fuel blend with other additives; and

(iii) using different fuels having more satisfactory antiknock and emission characteristics than gasoline.

In considering these options, one has to keep in mind their effect on the very large existing fleet of vehicles. Some may be new car technologies, others may require more or less expensive retrofitting, and yet others may be totally unsuited to the older but still useful car.

The Commission is surprised by the paucity of information on the health and environmental impacts of such changes; indeed, the available evidence deals almost solely with the regulated exhaust constituents--carbon monoxide, unreacted hydrocarbons and oxides of nitrogen--and whether they are within the limits prescribed. Most of the literature relates to the technical aspects of the fuels or blends, including effects on driveability, engine wear and corrosion, catalyst plugging and vapour pressure. Although the toxicity of the actual additives has been examined, there is little information on their combustion products, and the forms in which they reach the environment and target organisms, particularly humans.

Refining

Crude oils are separated by distillation into several progressively heavier fractions, whose properties depend on the size and structures of the different hydrocarbon molecules. These fractions are separated, treated and blended to make a variety of different products, such as gasoline, jet fuels, diesel and furnace oil (PACE, 1985). Generally the lighter (i.e., lower boiling) fractions that contain relatively short molecules (chains of up to 8 carbon atoms) are those used to manufacture gasolines. In order to increase the yield, however, heavier fractions with larger molecules are broken down or cracked into smaller molecules with lower boiling points.

Intermediate refinery products are upgraded into more effective fuels by reforming, a process that typically changes a low octane (60-70) material to about 90 road octane (RON) by increasing the proportion of aromatic hydrocarbons and converting hydrocarbon molecules with straight chains of 8-carbon atoms into molecules with branched chains.

Octane rating can be raised by more severe reforming, but this tends to increase the concentration of the aromatic hydrocarbons in the final product. Aromatic compounds typically constitute 20-35% of the volume of regular unleaded gasoline, and up to 50% of premium gasoline. The aromatics produced are predominantly xylenes and toluene, with smaller amounts of benzene. Several aromatic compounds, including benzene (USDH, 1982), are known carcinogens. Several are toxic. The health effects of these compounds are described below under additives.

All such aromatics increase the vapour pressure, i.e. volatility, of the gasoline blend, increasing evaporative emissions, the level of exposure for workers in service stations, and for the public in self-serve stations (US EPA, 1978 a). Although almost all the aromatic compounds are destroyed in the automobile catalytic converter, small amounts do escape in the emissions, and these quantities will increase with the higher concentration of aromatics in the gasoline. In view of this problem, and the large number of older vehicles without catalytic converters that will be on the roads for at least the next decade, more severe reforming alone does not seem to be a prudent approach to achieving the octane levels required.

Far more satisfactory is isomerization of the front-end naphtha, a low boiling fraction of low octane value, rich in straight chain molecules with predominantly 5 carbon atoms. The branched chain forms (isomers) of these compounds have very high octane values, and the isomerized fraction is blended back to produce a gasoline with the desired characteristics. Conversion requires a special isomerization plant. None has as yet been designed and built to operate in Canada for motor fuel (PACE, 1985), although they are common in the chemical industry, and for isomerizing butane in refineries.

Isomerization appears to be a most attractive option, since the exhaust emissions are those of gasoline. The health implications should be no greater than at present for efficiently functioning catalyst-equipped cars. For older cars, the lead content of gasoline can be reduced to the levels apparently needed to avoid valve recession (Weaver, 1984). The capital cost is high, but the extra cost to the consumer will be quite small (in the order of 1¢ per litre in the retail price of gasoline). The construction of isomerization capacity will also provide a short term boost to the engineering and construction sectors of the economy. As the isomerization plants will operate in Canada using Canadian materials, there will be no long-term drain on the balance of payments (RSC, 1985, pp. 37-45).

A cautionary note must be sounded, however, with respect to <u>all</u> gasoline fuels. A paper on the chronic toxicological properties of gasoline, published recently by the American Petroleum Institute, Exxon Research and Engineering Company and others (MacFarland et al., 1984), indicated that chronic low level inhalation of unleaded gasoline vapour produced a high level of renal carcinomas and sarcomas in rats, and histopathological examination revealed an increase in liver nodules at higher exposures. There is some evidence that the renotoxic effect of whole gasoline may be due to the presence of one or two of the five main groups of hydrocarbons present in gasoline, iso-alkanes being the most suspect. Work on this project is continuing, and the relevance of the results to human subjects is under active investigation. It should be noted, however, that typical human exposures to the concentrations used in the experiments are much shorter. Nevertheless, if this work is substantiated, the continued use of, or exposure to, gasoline fuels will have to be carefully evaluated.

Additives

Tetraethyl lead has been extensively used as an octane enhancer since the mid 1920's, though it has been progressively phased down since 1974 in North America. Its use in gasoline has led to widespread distribution of lead throughout the human environment (Nriagu, 1985), and its continued use has led to concern about health impacts. Regulations now restrict the use of lead in gasoline; other regulations will require all new light duty vehicles to be equipped with catalytic exhaust converters and use unleaded gasoline. As a result, lead will effectively disappear from fuels by the mid 1990's (RSC, 1985, pp. xiv). Several other additives have been suggested to replace lead as knock preventative and octane enhancer.

Methyl cyclopentadienyl manganese tricarbonyl (MMT) has been used extensively since 1957 as an antiknock replacement for tetraethyl lead. Developed and marketed by Ethyl Corporation, it is currently used in Canada in unleaded fuels (PACE, 1985). It remains in use in the U.S. in leaded gasoline in concentrations that have increased since the reduction of the lead content in that country, although it has been prohibited in unleaded gasoline because it is believed to interfere with effective reduction of exhaust emissions (Benson, 1978; Benson et al., 1979; US EPA, 1978 b, 1984). This does not seem to be the case with current technology vehicles (Shantora et al., 1985); indeed, in eight years of use of MMT in unleaded gasoline in Canada there does not appear to have been a higher incidence of catalytic converter failure than in the United States. MMT does not appear to cause failure of the oxygen sensor or deactivate the catalyst. The converters that were prone to plugging in the 1970's were of the monolithic type, consisting of a ceramic base impregnated with the precious metal catalyst. The pore size of these catalysts was much smaller than the modern pelletized catalysts which prevent the development of engine back-pressure.

The effects of MMT on automotive emissions are very small. They appear to range from slightly improved to slightly worse than for clear unleaded fuel, but it is unlikely that even a fleet test of unprecedented magnitude and scope would be large enough to show any statistically significant differences (Falkiner, 1986). The Canadian General Standards Board (CGSB) is currently reviewing the effects of MMT use on vehicles in Canada, and it expects to complete its study in mid-1986.

MMT itself has been found to be extremely toxic and to require stringent precautions at all stages of manufacturing, transportation and blending with gasoline. It penetrates the skin readily, and its ultimate site of action is the central nervous system. Studies with rats have shown that MMT is rapidly metabolized and distributed to the liver, kidneys and lungs, with the latter showing the greatest immediate toxic effects at low concentations (NHW, 1978). Its concentration in gasoline blends, however, is extremely low (0.072 g/L; 0.018 g Mn/L), so that the primary concern appears to be with its exhaust emissions. MMT is almost completely oxidized (99.7 %), mostly to Mn_3O_4 and MnO, with smaller amounts of other oxides present (Ter Haar et al, 1975). Any uncombusted MMT is rapidly photochemically decomposed.

It has been widely claimed that, as manganese is an essential component of human and animal diet, the anticipated levels of <u>atmospheric</u> manganese should pose no threat to human subjects (NHW, 1978, 1983, 1984; Cooper, 1984; Schroeder et al., 1966). However, this subject cannot be dismissed without investigating the possible accumulation in dusts and soils over extended periods of time, a process that has been responsible for the most intractable aspects of the lead issue.

Unlike lead particles, the manganese oxides that reach the soil are not likely to remain concentrated in the upper few centimetres for any length of time. The pH of the generally moist conditions prevailing in soils will cause mobilization of the manganese, which will move to lower levels and ultimately reach the groundwater or surface waters (Costescu and Hutchinson, 1972). Thus the manganese of gasoline origin actually reaching human populations will indeed be almost exclusively directly inhaled or ingested, in incremental quantities insignificant compared with the normal exposure through food and respiration (0.3 μg/day additional intake against an uptake of 120 μg/day without MMT) (NHW, 1978; Ethyl Corp., 1985). Average intake of manganese in Canada is about 3600 μg/day from food, 40 μg/day from water and 2 μg/day by direct inhalation.

Manganese and its compounds, particularly the chlorides and oxides, have been extensively studied in recent years, largely because of the fact that high exposures give rise to manganism, a toxic condition of the central nervous system that has symptoms similar to Parkinson's Disease. Ulrich et al. (1979) reviewed the conditions of exposure at which symptoms of manganese intoxication become apparent. They found these to be in excess of six months at levels above 2-11 mg Mn/m^3, although the albumin/globulin ratio was disturbed at six month exposures as low as 0.03 mg/m^3 and some evidence of peribronchial and perivascular sclerosis were noted at 0.3 mg/m^3. Manganese oxides and chlorides administered at higher dosages (up to 400 mg/kg body weight) affected phosphatase metabolism and led to an increase in calcium and degenerated neurons (nerve cells) in rabbits. Similar dosages depleted the dopamine content of the caudate nucleus in monkeys.

Ulrich and his co-workers then designed experiments to observe the effects of chronic exposure similar to those likely to result from internal combustion engines. His exposures were 100 to 10,000 times as much as the normal ambient atmospheric levels of manganese (1.5 $\mu g/m^3$ in the most heavily industrialized manganese-related centres and 0.1 $\mu g/m^3$ in others). They exposed rats and monkeys to levels of 11.6 to 1,152 μg Mn/m^3, generated as an Mn_3O_4 aerosol by burning MMT in propane, and did hematological and serum biochemical and histopathological evaluations at regular intervals. No clinical signs of toxicity were found in any of the animals, although at the highest levels (1,152 μg/ m^3) accelerated weight gain and slightly elevated hemoglobin were noted, as was some evidence of hypophosphatemia. No histopathological abnormalities were observed. Evaluations of pulmonary function, electromyographic activity, and limb tremor showed no effect; nor did tissue manganese levels change after six months. After nine months exposure, however, reversible elevated manganese was found in kidney, lung, spleen and blood; this was dose related.

The fact that liver manganese showed no increase is consistent with the homeostatic control of manganese metabolism through mitochondria. Manganese accumulates preferentially in tissues rich in mitochondria, but unfortunately, in contrast with the case of lead, little is known of its effects on cell biochemistry following chronic exposure to low levels.

Several papers have appeared describing manganese retention and distribution, many written by EPA scientists in response to the limited information available in the mid-1970's on the toxicity of Mn_3O_4 from MMT exhausts. It is now generally accepted that infant rats retain up to twenty times more manganese than adolescents and adults (Cahill et al., 1980; Kostial et al., 1978) and that manganese accumulation is promoted by iron deficiency in the diet (Lasky et al., 1982; Rehnberg et al., 1982). All the rats used in these experiments were fed manganese at rates in excess of 70 μg/day/g body weight, and as much as 3,550 μg/g, which are much higher than the Canadian average intake for a 70 kg adult (3,600 μg/day, equivalent to 0.05 μg/day/g). At these rather high levels of exposure homeostasis apparently does not occur in young rats before weaning, when blood-brain and blood-testicular barriers to manganese start to be noticeable, and the intestinal wall begins to prevent or control uptake. Elevated manganese concentrations in the brain have been observed to alter neurotransmitters, and Rehnberg et al. (1981) report that it is strongly absorbed in the cerebrum, hypothalamus and pituitary of pre-weanling rats. As it has a long residence time, these authors conclude that "... the neonate is sensitive to the toxic effects of Mn_3O_4", but no evidence of actual damage is presented.

Rehnberg et al, (1980) have also noted dose-related acceleration of post-natal liver iron depletion, depression of erythrocytes, hematocrit, hemoglobin and body weight and survival; all these experiments were done at manganese dosages between 21 and 214 µg/day. Bird et al. (1984) found that monkeys exposed to excessive (greater than 30 $\mu g/m^3$) manganese dust developed neurological abnormalities resulting from dopamine concentrations, which were related to the quantities inhaled and the period of exposure; but as in all the other work cited, these levels cannot be related to the chronic low level exposures of concern in the gasoline issue.

Barbeau (1986)* has suggested that it may be more important to compare the average additional intake of manganese predicted for MMT use (0.3 µg/day) with the present average intake due to inhalation (2 µg/day), than with the current daily uptake from food and water (100 - 140 µg, according to diet and location). Inhaled manganese will be absorbed in the lungs and some transported directly to the brain, probably exerting a more significant neurological effect than the ingested manganese uptake, which is first metabolized in the liver. These inhalation exposures, however, are two orders of magnitude below the lowest used in Ulrich's (1979) carefully controlled chronic inhalation study on rats (p. 8), at which no histopathological, pulmonary or electromyographic abnormalities were

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*We were sorry to hear of Dr Barbeau's death shortly after this discussion.

observed, although Bird (1984) did note the onset of dopamine-related abnormalities over the same general range of exposures.

Parkinson's Disease has been postulated to result from the interplay of environmental factors and individual genetic susceptibilities, against a background of normal ageing. Many potential toxins are detoxified by hydroxylation in the liver by P450 cytochromes. Barbeau and his co-workers (1985) have suggested that people with defective hydroxylation mechanisms may be more susceptible if exposed to environmental neurotoxins and, as a result, develop chronic degenerative disorders like Parkinson's Disease. Manganese is known to be implicated in parkinsonism (Barbeau, 1984; Cotzias, 1958), and Barbeau (1986) has suggested that it may also inhibit hydroxylation by P450 cytochromes. This might be particularly significant in sensitive gasoline station attendants if MMT were to be used.

MMT has already been used, however, for 8 years in unleaded gasoline, which currently comprises about half the gasoline consumed in Canada. The additional exposure to manganese is well within the normal range represented by dietary variations, and is likely to remain so. Aside from the occupational problem related to the possible genetic susceptibility of some workers in gasoline stations, Cooper's view (1984) that the general public has a wide margin of health safety with respect to the worst case use of MMT in gasoline appears to be sound.

<u>Aromatic Hydrocarbons: Benzene, Toluene and Xylenes</u>. Many aromatic hydrocarbons are toxic, and some are carcinogenic to both humans and experimental animals (NHW, 1979; IARC, 1982; Maltoni, 1983; Goldstein, 1983). Of concern is whether their increased use as gasoline additives might pose any hazard to the general public.

Benzene is quite widely used in Europe as an octane enhancer in gasoline, and the European Communities allow a maximum level of 5% in the fuel (CEC, 1985). Benzene is not added to gasolines in North America, and there is no maximum permitted level in Canada or the United States, but the proportion used is generally about 2-3% in the final blend. In some countries it may be as high as 15% (Maltoni, 1983). The reforming process produces large amounts of benzene, toluene and the three molecular forms (isomers) of xylene in the refinery (PACE, 1985). Significantly higher concentrations of aromatic compounds are found in all gasolines refined from tar sands.

Despite the fact that benzene is a good octane enhancer, it commands a high price as a chemical feedstock for the manufacture of plastics, and most of it is removed. The chemical demand for toluene and xylenes is smaller at present and, being quite efficient octane enhancers, they are left in the reformate, constituting between 15 and 20% of the final gasoline blend (Halpern and Noble, 1985). This situation is likely to end abruptly once the disappearance of lead banking allows the full impact of the rapid phasedown of lead in the U.S. to be felt in the marketplace, probably in 1988.

Halpern and Noble (1985) believe that, had the U.S. lead reduction been planned over a period of five years or more, refiners would have had the time to make up the overall pool octane shortage of some 2.5 octane numbers through plant investment in such processes as ultra-high severity reforming, alkylation or isomerization. The rapid phasedown restricted the options open to refiners, allowing them only to increase the reforming severity of their existing plant or to blend in aromatics; industry capacity for the manufacture of MTBE and tertiary-butanol-methanol octane enhancers is currently inadequate to make up the entire shortfall. Increases in reforming severity will recover about half the octane shortfall, largely by increasing the proportion of aromatics. The remainder will have to come from further aromatic additions.

As aromatics become more valuable and there is increasing competition between the petroleum and chemical industries, their value as octane enhancers will rise relative to that as chemical feedstocks, leading to competition for their supply, and inevitably to higher prices. Furthermore, the limited volume of aromatics that will be available within the U.S. will have little effect on the octane shortfall, so that imports will be needed until the investments in new plant processes can redress the balance in the market (Halpern and Noble, 1985).

Refineries in Canada generally produce gasolines containing about 2% benzene (rarely exceeding 4%). The median total aromatics seems to be in the vicinity of 20%, although a few are already approaching 50% in order to achieve acceptable octane levels (NIPER, 1983). Although Canadian refiners, unlike their U.S. counterparts, are permitted to use MMT in unleaded gasoline, it is not as effective an octane enhancer as tetraethyl lead, and we shall also probably face greatly increased proportions of aromatic hydrocarbons in gasoline.

Aromatic additions increase the vapour pressures of the fuels, leading to evaporative losses to the atmosphere from the fuel system during driving. This fact has already resulted in regulations mandating the venting of carburetors and fuel tanks through canisters filled with activated carbon. Elevated vapour pressures are also responsible for vapour lock in the fuel system and other driveability problems, and to greater exposure to fuels through inhalation and handling in filling stations (US EPA, 1978 a). Though this might be expected to represent mainly an occupational hazard, the increasing number of self-serve gasoline stations exposes an ever-larger proportion of untrained members of the general public; exposure levels in gasoline stations are similar to industrial exposures (NHW, 1979).

Benzene is known to be both a toxin and a carcinogen; many of its toxic properties were documented during the 19th century, and its carcinogenicity was first reported by Delore and Borgomano (1928). Its main exposure route is through inhalation. The US OSHA (1985) standard is 3.2 mg/m^3 for 8 hours, and the action level is 1.75 mg/m^3. Although levels of 10,000 mg/m^3 can be tolerated for 30 to 60 minutes, acute effects become apparent at exposures above 3,500 mg/m^3. Acute symptoms generally involve the central nervous system--muscle tremors, convulsions and paralytic asphyxiation, which in non-lethal cases can persist for several weeks after the event (NHW, 1979).

Humans absorb about half the benzene inhaled, and retain about 30%, the remainder being exhaled unchanged (NHW, 1979; IARC, 1982). The retained benzene is metabolized mainly in the liver, where it increases lipid peroxidation (Khan et al., 1984), and other lipid-rich sites such as bone marrow where much of the synthesis of blood occurs. Its effects on the hematopoietic system are thus not surprising. The hemotoxicity of chronic exposure mainly results from destruction of the myeloid and erythroid components of the bone marrow, leading ultimately to pancytopenia, in which there are decreases in white blood cells (leucopenia), red cells (anemia) and platelets (thrombocytopenia) (Goldstein, 1983). There is consequently a decrease in the body's ability to resist infections. Blood cell chromosome abnormalities have also been reported (Snyder, 1984). Little is known of the mechanisms of the processes, but some of the metabolites have been identified with radioactive tracers. The most important are phenol, catechol, quinol and hydroquinol; the specific effects of benzene toxicity are believed to be mediated by benzene epoxide, which is a transient metabolic intermediate (Irons, et al., 1983), although a toxic aldehyde precursor of muronic acid has also been suggested (Gad-El Karin et al., 1985).

These hematological effects have been reported at occupational exposures of 17.5 to 122 mg/m^3 for three months, although they occur more generally at exposures above 350 mg/m^3 (NHW, 1979; Snyder, 1984). Age, sex and familial factors are believed to play a role in individual susceptibility to pancytopenia. Removal of the exposed worker usually results in recovery, although some effects may persist for several years (NHW, 1979; Sato et al., 1975; Aksoy et al., 1974, 1976).

There is strong evidence for a relationship between benzene exposure and some forms of leukemia, although proof of this has been hampered by the lack of an adequate animal model upon which to base a dose-response relationship. Also restrictive are the absence of satisfactory epidemiological data, which are almost impossible to obtain, and the incomplete understanding of the mechanism of induction (NHW, 1979). Other hematological disorders that may be related to benzene exposures are Hodgkin's disease, lymphocytic lymphoma, myeloid fibrosis and multiple myeloma.

Goldstein et al. (1980) observed a few cases of acute and chronic myelogenous leukemia in benzene-exposed rats and mice, and suggested a possible causative effect, but Maltoni (1983) asserted that their data were inadequate. Maltoni was able, however, to use an animal model satisfactorily to induce zymbal gland carcinomas in rats and demonstrate a dose-response relationship. Goldstein (1983) considers benzene to "... definitely ... be a cause of leukemia in man". He derives his evidence from a number of different approaches, different types of epidemiological studies and many case reports. Goldstein refers to the impressively large numbers of case reports associating benzene with acute myelogenous leukemia and the frequency with which benzene-induced pancytopenia has been followed through a pre-leukemic phase into acute myelogenous leukemia. Table I summarizes Goldstein's view of the current state of knowledge of the relationship of benzene exposure to hematological disorders.

Urban non-occupational exposure of individuals to benzene has been estimated at about 125 mg/yr, of which 90 mg comes from food, but the significance of oral against respiratory uptake cannot be evaluated (NHW, 1979). Ambient air exposure is in the range of a few micrograms per cubic metre, of which 80% owes its origins to gasoline-related emissions, although Weaver et al., (1983) found atmospheric benzene levels in pristine areas to be about 60 $\mu g/m^3$. At the levels encountered in the atmosphere, there is no clinical, experimental or epidemiological evidence that points to demonstrable health effects in humans, but the Department of National Health and Welfare cautions that this chronic exposure is spread over a lifetime, so that there may be <u>some</u> effects, so far unidentified (NHW 1979). Indeed, if there were a major increase in benzene in gasoline, the population exposure to benzene would undoubtedly be much higher than the department estimated in 1979.

Women are especially at risk from benzene due to its high liposolubility, and elimination from the body is likely to be slower due to their higher fat/body weight ratios (Sato, 1975). Their hormonal balances are

Table I

Relationship of benzene exposure to various hematological disorders.

A. Causality Proven

1. Pancytopenia: Aplastic Anemia
2. Acute Myelogenous Leukemia and Variants (Including Acute Myelomonocytic Leukemia, Acute Promyelocytic Leukemia, Erythroleukemia)

B. Causality Suspected

1. Chronic Myelogenous Leukemia
2. Chronic Lymphocytic Leukemia
3. Hodgkin's Disease
4. Paroxysmal Nocturnal Hemoglobinuria

C. Association Suggested But Unproven

1. Acute Lymphoblastic Leukemia
2. Myelofibrosis and Myeloid Metaplasia
3. Lymphoma: Lymphocytic, Histiocytic
4. Thrombocythemia

Source: Goldstein, 1983

also likely to be more severely affected (NHW, 1979). The placenta is not a barrier to volatile solvents, but fetal exposures to levels not toxic to the mother do not appear to produce teratogenic effects (Lee et al., 1983).

Many aromatic hydrocarbons have been shown to be carcinogenic to animals, and some to humans (USDH, 1982). The group of polyaromatic hydrocarbons (PAH) is particularly dangerous, but they are present in gasoline exhaust emissions in only minuscule quantities. The most significant of these compounds is benzo-α-pyrene, which may constitute as much as 0.0003% of the exhaust gas; PAH's are, however, of greater concern in diesel exhausts. Most of the benzene and other aromatic hydrocarbons in gasoline are undoubtedly oxidized and destroyed during the combustion process and the subsequent catalytic conversion of the exhaust gases (Nebel, 1979), although some benzene is actually produced in the fuel combination process from precursors present (Black et al., 1980). The extent to which unconverted aromatics escape, especially through malfunctioning converters, remains uncertain and is masked by emission standards that generally refer to total hydrocarbons and do not discriminate further. Black et al. (1980), however, found that as total hydrocarbon emissions were reduced, the proportion of paraffins in the exhaust increased; i.e., the proportions of aromatics decreased. This implies a more efficient removal of aromatics from the exhaust gas than paraffins.

Some recent work on benzene emissions corroborates this assertion (Seizinger et al., 1986). Two groups of fuels were made, one containing 25% aromatics, and the other with 40%. Each group comprised three fuels in which the benzene concentrations were 0.03%, 1.5%, and 4%. Unfortunately, the individual concentrations for the other aromatic hydrocarbons in the fuels were not specified. The exhaust and evaporative emissions of benzene and total hydrocarbons were measured, and are here expressed in grams per kilometre driven. Only exhaust emissions are, of course, quoted for CO and NO_x. Increasing benzene concentration led to a small increase in benzene exhaust emissions, although total hydrocarbons decreased slightly. The combustion process and the catalyst were seen to be remarkably efficient in removing benzene; a 100-fold increase in the concentration of benzene in the fuel, together with some small amount of benzene actually formed during the combustion process, resulted in less than doubled benzene exhaust emissions. For the 25% aromatic base fuels the measured benzene exhaust emissions increased from 5.0 mg/km to 8.7 mg/km, and hydrocarbons decreased from 0.22 g/km to 0.18 g/km. For the 40% base aromatic fuel, hydrocarbons showed no change with benzene concentration. Evaporative emissions for benzene showed little difference with concentration but hydrocarbons are distinctly lower in the 40% aromatic base fuels.

The health effects of toluene and xylene have been reviewed for the Department of National Health and Welfare (NHW, 1985) and by the US Environmental Protection Agency (1980). Toluene is the most abundant aromatic hydrocarbon contaminant in air, the average concentrations of toluene, o-xylene, m-xylene and p-xylene in the Los Angeles Basin being respectively 151, 38, 76 and 28 $\mu g/m^3$. Pilar and Graydon (1973) showed atmospheric toluene and benzene contamination in Toronto to be closely linked with automotive traffic density. All these aromatics have been found in Canadian drinking water, generally in microgram per litre concentrations.

The main route of exposure of populations to toluene is by inhalation, with smaller quantities ingested through water. Exposure of Canadians is estimated to be about 2.3 mg/person/day on average; the worst situation is in Toronto (141 mg/person/day). Mean xylene exposure is about 0.95 mg/person/day. About 40% of the intake of these compounds is immediately exhaled and between 70% and 90% of the amount retained is rapidly metabolized and eliminated, with between 3% and 20% exhaled unchanged.

Inhaled toluene is rapidly transported by the blood to lipid tissues but ingested toluene has first to be metabolized by the liver before reaching the central nervous system. Toluene is metabolized by a mixed function oxidase system to benzyl alcohol, which is oxidized in turn to benzaldehyde and benzoic acid and then conjugated with glycine to form hippuric acid; about 80% of the toluene retained is excreted in this form in the urine within 24 hours. Xylenes behave in much the same way, 95% being excreted as methylhippuric acid. The only danger of accumulation seems to be in adipose tissues on repeated exposure.

Extremely large doses (1 g/kg body weight) are required to show mutagenic effects such as chromosome damage in rat bone marrow. In vitro studies on humans showed no evidence of mutagenicity; no significiant chromosomal aberration was detected in workers exposed to toluene, some exposures being as high as 200 ppm for 15 years.

There have been few studies on carcinogenicity of these toluene-derived compounds. Though they are not themselves carcinogenic, they may be co-carcinogens when inhaled. Exposure to toluene at 300 ppm for 24 months produced no increased neoplastic, proliferative or degenerative lesions in rat organs, and there are no indications that xylene is carcinogenic in animals. In humans, occupational exposure to aromatic hydrocarbon solvents has been linked with lymphatic leukemia, but this cannot be related to any one of these compounds.

Toluene and xylene have been reported to have some teratogenic effects when administered to rats and mice in very large doses--about 0.2 - 0.7 g/kg body weight, and on inhalation at 1 g/m^3. Both are considered to be slightly to moderately toxic, with LD_{50} values for rats ranging from 3.5 to 5 g/kg. These large doses generally affect the central nervous system, leading to dose-related alterations, such as fatigue, confusion, headache and nausea. All have narcotic effects and cause eye irritation. Recovery is quite rapid on termination of exposure. An investigation of rats showed no effects in blood, urine or tissue samples between 30 and 1,000 ppm of toluene vapour; some adverse central nervous system disorders and serum cholinesterase irregularities were noted in rats exposed to xylene vapour (15 mg/m^3) for 85 days.

Emissions from cars fitted with efficient catalytic converters will be low, given the low levels of observed total hydrocarbons. Without reliable information on the toxicity and carcinogenicity of toluene and the xylenes at the very low ambient concentrations likely to be experienced, it is difficult to assess the health risk faced by the population, although it is almost certain to be low. Nevertheless, in the face of possible increases in the aromatic hydrocarbon contents of severely reformed unleaded fuels, it would surely be prudent to regulate the level of benzene and minimize the presence of other aromatic compounds in gasoline. The presence of aromatic hydrocarbons--particularly polyaromatics (PAH's)--in diesel fuels and exhausts is also a matter of great concern that should be further investigated.

Methyl Tertiary Butyl Ether (MTBE). Use of this octane enhancer at concentrations up to 7% in unleaded gasoline is allowed in the United States under an EPA waiver granted in 1979 (EPA, 1979). MTBE is made from methanol and isobutylene (Chase & Woods, 1979), and is totally miscible with gasoline, since it has similar physical properties to the alkanes. The refining industry has seen this as a way of increasing octane by adding a methanol group to gasoline without having to use a cosolvent (Penny, 1983) although its manufacture imposes a substantial energy penalty (Colledge, 1986; COFA, 1985).

In one evaluation, the fuel properties of MTBE and MTBE-gasoline blends were examined, and engine studies showed power output and energy efficiency to be virtually the same as for gasoline fuels (Johnson and Taniguchi, 1978). Exhaust emission tests showed CO to decrease substantially with increasing MTBE, and NO_x to increase somewhat, while hydrocarbon emissions were essentially unchanged. Furey and King (1980) found that when evaporative emissions were reduced by a closed loop carburetor, there was little difference between emissions from MTBE blend and gasoline; without the closed loop, MTBE blend emissions were lower. Aldehyde emissions increased slightly, depending on the fuel/air ratio.

Investigators concerned with safety and toxicological aspects of MTBE have found that MTBE is "...no worse than gasoline" in terms of effects due to inhalation, ingestion or skin absorption (Reynolds et al., 1974) and that it "... is relatively unreactive in the atmosphere, with little tendency to form O_3". Evaporative emissions for 15% MTBE gasoline blends may be as much as 15% higher than for gasoline, while cold start driveability is worse than for gasoline (Furey and King, 1980). No exhaustive study of the toxicology of MTBE or its combustion products appears to have been undertaken, but it could have a positive health effect by obviating the need for severe reforming with its consequence of increased benzene and other aromatic hydrocarbons.

Major investments have recently been made in Saudi Arabia, Finland, Italy and Ireland to manufacture MTBE (Oil and Gas Journal, 1984; Hydrocarbon Processing, 1984; Chemistry in Britain, 1985; European Chemical News, 1984). Although Canada is one of the world's largest producers and exporters of methanol from natural gas, isobutylene is in short supply in this country, particularly in western Canada where most methanol is made, and the manufacture of MTBE may not prove to be economically feasible with present technology. If MTBE is used to replace lead antiknock agents, it is likely to be imported, which fact, for the large quantities foreseen (up to 15% of the total volume of gasoline consumed in Canada), will have a significant negative impact on the balance of payments.

Alcohols. The use of alcohols as blends with gasoline or as alternative fuels has been the focus of a great deal of discussion in recent years. The possibility of reducing dependence on imported oil while at the same time producing feedstock from renewable (i.e., agriculturally produced) biomass has attracted the attention of government and populace alike. Alcohol blends are extensively used in many countries--ethanol in Brazil, gasohols (gasoline/alcohol mixtures) in many parts of the United States, methanol in Germany. One supplier is marketing an ethanol blend in Manitoba, a methanol-ethanol blend in Saskatchewan, and plans to extend the sale of the latter into Alberta. Another major petroleum marketer is test-marketing a methanol-butanol blend in Ontario.

The House of Commons Standing Committee on National Resources and Public Works is currently considering the implications of widespread use of alcohol fuels in Canada. For Canada, the options appear to be:

(1) gasoline/methanol blends with ethanol or TBA as cosolvent-- Canada is one of the world's largest producers of methanol from natural gas and has surplus capacity; and

(2) neat methanol, which is really an alternative fuel, and is discussed later (see section on alternative fuels).

There is an extensive literature on the technical problems of using methanol blends (5 % methanol) in engines and systems designed for gasoline (MVMA, 1985). These range from corrosive effects on the terne plate of gasoline tanks, to chemical reactions with engine components and hoses, particularly plastics. Methanol is more soluble in water than in gasoline and, unless a cosolvent such as ethanol or t-butanol is used, the absorbed moisture can cause separation into two liquid phases (gasoline and a methanol-water solution), with disastrous results for fuel combustion, efficiency and driveability.

Methanol, if simply added to normal gasoline, would produce a higher fuel vapour pressure than gasoline blended in accordance with Canadian General Standards Board specifications. Refiners who use methanol in gasoline blends must compensate for the higher vapour pressure of methanol by removing some of the lighter hydrocarbons, mainly butane, that are normally blended into gasoline. Butane is a low cost, relatively high octane hydrocarbon with higher energy content than methanol. The substitution of methanol for butane, therefore, carries a levered economic debit that reduces the apparent octane cost advantage of methanol. In provinces where gasoline does not have to meet the CGSB vapour pressure specification, methanol blends may become more attractive.

Switching between different blends or between blends and straight gasoline poses problems for existing vehicles. Though some of the automotive manufacturers have produced experimental engines that can cope with switching throughout the entire range between gasoline and neat methanol by using modified fuel injection systems, this is essentially new car technology and therefore a longer-term option (MVMA, 1985). Almost all the literature on alcohol/gasoline blends relates to their use and effectiveness in conventional vehicles. Some papers and reports deal with the emissions of regulated pollutants (CO, hydrocarbons and NO_x). Only a few are concerned with non-regulated emissions (primarily aldehydes), and not one was found to deal extensively with the health implications of widespread use of alcohols as or in automotive fuels.

There do not, however, appear to be significant differences in the composition of the unburned hydrocarbon emissions between methanol blends and the unblended gasoline-- n-butane, toluene, iso-pentane and other volatile gasoline constituents that dominate the exhaust emissions (Gabele et al., 1985).

Others have drawn attention to the importance of the air-fuel ratio in achieving low emissions with alcohol blends (MVMA, 1985; Wathne and Hov, 1985). Where the activated carbon canister used to prevent evaporative emissions from the fuel system and the carburetor fails (canister break-through), n-butane comprised about 70% of the total evaporative emissions, compared with 25% otherwise (Gabele et al., 1985). Only barely detectable quantities of methanol were present in a relatively small percentage (15%) of Gabele's tests on blended fuel. The blends used in the research, however, tended to have higher vapour pressures than the straight gasolines as they were simply mixed and not blended, and the volatile butane content was not reduced (or backed out) to conform to any particular specification.

In methanol blends, carbon monoxide and hydrocarbon emissions are found to be generally much the same as for straight gasolines, while oxides of nitrogen are somewhat lower (Gabele, 1985). Methanol is, however, a primary alcohol, and like all members of this group, is oxidized to form an aldehyde, in this case formaldehyde; similarly, using ethanol as cosolvent would lead to acetaldelyde as well. Aldehyde emissions are indeed found to be some 50% higher with the blends than for straight gasolines (Gabele, 1985), but the amounts involved are extremely small and they are quite efficiently removed by the catalysts, so that the possibility of negative health effects appears to be remote. The aldehyde situation for neat methanol fuels (85-90% methanol) is somewhat different (see below).

Alcohol blends present an attractive health and environmental alternative to the increase of aromatic hydrocarbons resulting from severe reforming.

Alternative Fuels

Six practical fuel systems exist for automotive propulsion: gasoline, diesel, alcohols (neat methanol in Canada), propane, compressed natural gas (methane), and electricity. All have advantages and disadvantages, and some have limitations that at present restrict their use to specific types of vehicles and functions. The massive world-wide investment in the manufacture and use of traditional gasoline vehicles is a powerful determinant of the economics of alternative power systems. The success of these vehicles was for many years responsible, in large measure, for the lack of interest in research and development on alternative power systems--high energy-density batteries, for example. It is therefore not surprising that four out of the five alternatives to gasoline are themselves products of the petroleum and gas industries, and that these are the ones that require the least radical technological changes to the conventional gasoline propulsion system.

After a brief spell of popularity for light vehicles following the oil supply problems of the mid-70's, diesel fuel is once again used almost exclusively in heavy duty vehicles and cannot really be considered a replacement for gasoline. Were it ever to regain widespread popularity for light vehicle use, the health and environmental impacts of its aromatic hydrocarbon and particulate emissions would have to be carefully assessed.

Electric propulsion will remain a viable option only for fleets of short-haul local delivery vehicles large enough to carry the heavy lead-acid batteries that are presently the only practicable power source (Energy Ontario, 1982). This situation will not change until some of the recent exciting results of research in new, lightweight high energy-density systems can be developed into usable technologies. Electric systems are certainly the most benign from both environmental and health points of view because almost all the emissions are shifted from mobile to more easily controllable stationary sources--the power station providing off-peak electricity to recharge batteries in the home. Substantial use of electric power is unfortunately unlikely for many years.

Methanol as a Fuel. Canada is one of the world's largest producers of methanol and probably its largest exporter; only 14% of the production is actually used in this country (COFA, 1985). It is produced mainly in Alberta from natural gas, although there are also plants in Quebec. As new plants have come on stream in recent years, mainly in third world countries with low wage-cost structures and, more importantly, artificial exchange rates, the manufacturers are threatened by world oversupply and are seeking to expand the domestic market. The three automotive fuel options open to them are :

(i) gasoline blending, using ethanol or tertiary butanol as a cosolvent;

(ii) conversion to methyl tertiary butyl ether (MTBE);

(iii) using neat methanol.

From the manufacturers' point of view, neat methanol (90-95% methanol with 5 or 10% gasoline) is the preferred option as it does not requre a cosolvent, and it has the potential for the greatest sales volume. Production of MTBE for use as a gasoline additive is less attractive ecomomically due to the shortage of isobutylene, the other essential feedstock, in Western Canada. The Motor Vehicle Manufacturers Association (1985), in a brief to the House of Commons Standing Committee on National Resources and Public Works, recently clearly expressed their preference for neat methanol over blends as a third fuel option, complementary to gasoline and diesel. The MVMA views blends as being of value only in an interim period while substantial numbers of older cars are still on the roads, and then only under strict specification, as many of the methanol blend problems have resulted from inappropriate components or proportions. Neat methanol fuel requires new car technology. The existing fleet of vehicles was designed and built for gasoline, and many of the materials used are corroded by or unsuited to methanol. Design changes involve, inter alia, modified fuel injection systems and catalytic converters that will cope with emissions of methanol and its oxidation product formaldehyde, both of which are toxic.

Unlike gasoline, methanol combustion produces quite low levels of hydrocarbons, which are now regulated primarily for their role in the production of ozone, a powerful photochemical oxidant (Haagen-Smit, 1952). Methanol fuels do produce emissions with quite high levels of unburned methanol, which is not photochemically reactive, and about ten times more formaldehyde than is the case for gasoline (Gabele, 1985). Although formaldehyde is photochemically reactive, the quantities are very small, so that methanol fuels contribute less to the oxidant problem than does gasoline (Carey, 1981). Overall, however, the emissions of methanol and formaldehyde are together more or less equivalent to the hydrocarbon emissions from gasoline, and methanol produces about the same amount of carbon monoxide. Nitrogen oxides emissions are substantially lower for methanol (Wathne and Hov, 1985), and these could be reduced even further using three-way catalysts; alternatively, the reduction catalyst might be eliminated (Wilson, 1984).

The US EPA (Harvey, et al., 1984) has used a model developed by the Southwest Research Institute to relate methanol and formaldehyde emissions (in grams and milligrams per mile, respectively) to equivalent ambient concentrations (in grams per cubic metre), under typical and severe road traffic conditions in street canyons, roadway tunnels, expressways and garages. These are then related to a range of concern identified from what they call "... known toxicological and health effects" in order to define a set of design targets. These effects are taken from the literature on occupational health and safety and relate to the onset of clinically identifiable symptoms, such as irritations of the eyes, ear, nose, throat and skin as the lower limit for formaldehyde (0.03 mg/m^3). For methanol, the lower limit is 4.5 mg/m^3, the level at which dilation of the pupil of the eye in response to darkness is sensibly affected.

When actual emission measurements are compared with the equivalent ambient concentrations derived from the model (Harvey et al., 1984), only severe conditions in roadway tunnels would exceed the lower limit of concern for formaldehyde (0.03 mg/m^3), although garages might pose problems, especially in winter. Methanol emissions would not exceed the lower limit of concern (4.5 mg/m^3) under moving traffic conditions, but severe situations in garages would be above the limit. Vehicles with malfunctioning catalytic converters would only exceed the lower methanol limit in the severe roadway tunnel situation, but might exceed the intermediate point for formaldehyde (0.15 mg/m^3) under these conditions. No emissions are likely even to approach the upper limits of concern, which were chosen as the U.S. Occupational Safety and Health Administration proposed standards for workplace exposure--260 mg/m^3 for methanol and 1.3 mg/m^3 for formaldehyde).

Levels of formaldehyde in both personal garages and parking garages would have little effect on the user as exposures would be for short periods--generally only a few minutes; but workers in the building might well be subject to some degree of risk. The same might apply to children living in the vicinity of service stations and parking garages (Ontario

Ministy of Health, 1985). This work has been corroborated by Kraft and Kuhler (1985), who developed a similar model in Germany.

The toxic properties of methanol are well known, and it would have to be denatured, probably by adding 5-10% gasoline before it could be widely sold (Wathne and Hov, 1985). If ingested, it causes permanent damage to the central nervous system, usually blindness, and is often fatal. Little is known about low level chronic exposure, except that it appears to be mediated through formaldehyde by a liver oxidase enzyme. It is interesting that the first clinically noticeable effect should be on the eye at occupational exposures of 4.5 mg/m^3, but is not known whether the reduced ability of the pupil to adapt to darkness and changes in cerebral cortex reflex activity (noted at 1.2 - 1.5 mg/m^3) does in fact represent adverse physiological effects. What is clear, however, is that there are effects of methanol on the central nervous system at levels well below the occupational Threshold Limit Values (TLV).

The background ambient concentration of formaldehyde is approximately 0.1 - 0.5 µg/m^3, measured at sea and in other remote areas. It is produced by natural processes, such as atmospheric photochemical reactions, and from anthropogenic sources, mainly combustion processes. Formaldehyde makes up between 50% and 80% of all atmospheric aldehydes (Wathne and Hov, 1983). In the atmosphere it decomposes rapidly, so that it is not transported over long distances. Formaldehyde is a toxic compound suspected to have carcinogenic properties; its effect on human health at elevated exposure levels have been well described. Unfortunately the few studies of chronic long term exposure have been marred by uncertainties introduced by the presence of other air pollutants (Meek, et al., 1985).

Formaldehyde is a powerful irritant of the upper respiratory tract, particularly the nose and throat at concentrations in air as low as 1 - 2 mg/m^3; it has also been observed to halt all ciliary movement in the air passages of guinea pigs at this level. Formaldehyde is absorbed through the nasal mucosa, and on ingestion it has been reported to spread through the entire bodies of rats and mice within 5 minutes (Buss et al., 1964). The eye is particularly sensitive, with irritation reported at levels as low as 0.012 mg/m^3, and dose-response relationships measured down to 0.36 mg/m^3. It is classed as a strong allergen but, although cases of asthma and asthmatic bronchitis have been reported, allergic dermatitis is a more common reaction to formaldehyde sensitization (Wathne and Hov, 1985).

Although the Ames test for genetic mutation has shown no effect on Salmonella typhimurium, the bacterium used as a reference (Wathne and Hov, 1985), experiments with human cells in vitro have shown formaldehyde damage to human cells. Mutagenic effects have been reported on yeasts, bacteria, plants and insects (Magana-Schwencke et al., 1978). Formaldehyde is therefore classified as a weak mutagen. No teratogenic effects have been positively identified.

Suspicion of formaldehyde's carcinogenicity rests on exposure of rats and mice at concentrations down to 0.25 mg/m^3 for 24 months. Two out of 235 rats developed nasal carcinomas at 7.4 mg/m^3, and 103 out of 232 developed carcinomas at 18.5 mg/m^3. No cases were observed among rats at 2.5 mg/m^3. Only 2 mice developed neoplasms even at the highest exposures (Swenberg et al., 1980). The interpretations have been strongly criticized as the condition may have been precipitated by chronic tissue changes resulting from nasal lesions (Wathne and Hov, 1985). Despite several reports of possible formaldehyde-induced nasal and lung malignancies, the results do not provide an adequate basis for asssessing cancer risks. Nevertheless, the U.S. Occupational Safety and Health Administration (1985) has recently promulgated two proposed occupational exposure standards, 2.0 mg/m^3 as an irritant, and 1.3 mg/m^3 as a carcinogen, based on a daily 8 hour exposure. The intervention levels are 1.0 mg/m^3 and 0.67 mg/m^3 respectively.

There is thus need for research to identify the potential effects of methanol and formaldehyde at the low levels to which the public may become exposed <u>before</u> major investments are made in the production of neat methanol fuels and cars.

<u>Propane-Fuelled</u> <u>Vehicles</u> have long been used for short haul specialized purposes, such as fork-lift trucks, as well as stationary power sources. Unlike gasoline, which contains some 400 constituents, the fuel is almost pure propane. Its low cost has made propane-fuelled cars and trucks increasingly popular in recent years as taxis and local delivery vehicles. Fleet owners often maintain their own fuel filling stations, particularly in areas where bulk propane is not widely used. Low crude oil prices provided little incentive for vehicle manufacturers to develop engines suited to alternative fuels, so that conversion from gasoline to propane has been left to propane equipment manufacturers and aftermarket retrofitters. Propane-fuelled vehicles are generally retrofitted gasoline models that were not originally designed to run on this fuel, a situation that voids the manufacturer's warranty. The conversion industry is dominated by small firms, and there have been several reports of fly-by-night operators.

For optimum results, the engine should be designed with the fuel in mind, so that the buyer cannot expect comparable durability and performance from a modified version running on an alternative fuel. Drivers seem to be enthusiastic about propane-fuelled vehicles' apparent economy of operation, but there are many serious problems related to their operation. These have been reviewed for the Transportation Development Centre by Chrysler Canada Ltd. (Lacy et al., 1984).

Propane engines use rather simple carburetors based on the gasoline carburetor design. These are far from optimum with regard to fuel economy, power output and driveability. Unfortunately, good cylinder to cylinder fuel distribution is not assured by feeding in the propane as a gas, using the technology borrowed from gasoline for liquid fuels. The air/fuel ratios are critical to satisfactory operation, and the orifice temperature and pressure have to be carefully balanced against the exhaust manifold pressure to avoid knock. This has to be set mechanically, which is difficult for the often ill-equipped small retrofitter, so it is set at one point, usually 2,000 rpm. This produces high temperatures under other operating engine conditions, which in turn leads to rates of valve recession greater by a factor of 5 to 10 than is the case for an engine designed for unleaded gasoline. Unlike gasoline engines, spark knock occurs at higher engine speeds, where the sound is masked by other engine noise and thus cannot be corrected before damage occurs. The best retrofit systems are deficient at temperatures below -15°C, and some even at 0°C, although a block heater can extend the practical limit to -40°C.

Exhaust emissions have lower levels of hydrocarbons and carbon monoxide than do gasoline, but higher oxides of nitrogen, perhaps on account of the higher operating temperatures. They do not use catalytic converters, although some of the major car manufacturers believe that they should be fitted.

Propane is continuously vented to the atmosphere during refuelling, losing about 50 g per fill-up, typically twice a day for a taxi. Propane vehicles now account for only about 10% of the new fleet, but, since they tend to be heavily used, they probably emit as much total hydrocarbons as the rest of the fleet. There are currently about 125,000 propane-fuelled vehicles in the major metropolitan centres of Canada. The authors of the report believe that concerns about propane safety stem from unfamiliarity, and that they are safer than gasoline powered vehicles in crash situations. (Lacy et al., 1984).

Regulated emissions (carbon monoxide, hydrocarbons and oxides of nitrogen) are lower for propane than for gasoline. Unlike gasoline, these emissions are almost constant at temperatures above 0°C, but they tend to rise at lower temperatures. Carbon monoxide emissions are particularly low; hence propane's popularity as the fuel for fork-lifts intended for indoor use. There is little information on exposure to propane, other than the comment that it is an asphyxiant that excludes oxygen from the lungs. Once again, there are no details on the health effects of long term chronic exposures, nor of unregulated combustion products in the exhaust. So that its presence in air can be easily detected, small amounts of mercaptans are added. These are mildly toxic sulphur compounds with offensive odours.

<u>Compressed Natural Gas</u> (CNG) has both advantages and disadvantages when used as an automotive fuel. It would be both cheap and easily available, as well as providing a market for natural gas, especially during the summer months. Because it is a gas at ordinary temperatures and

pressures, it must be contained under pressure in the vehicle, necessitating heavy storage containers that require care in replenishing. Ideally, vehicles could be connected to household (low pressure) natural gas supplies overnight and pumped into the car storage bottles overnight; conventional filling stations would find it difficult to pump the gas to high enough pressures sufficiently fast to avoid massive line-ups., The capital cost of a single compressor station is high (about $250,000), so that this technology is better-suited to fleets, bringing it into competition with propane.

One advantage is that it is a new car technology. Retrofitting is not practicable, given the high pressures involved, which require computer control of feed and ignition. On the other hand, it is more difficult to ignite the fuel because its oxidation involves breaking the strong bond between the carbon and hydrogen atoms in the methane molecule. This ignition problem will probably require a dual fuel system to start the engine. There is also a 10-30% loss of power inherent in the system due to displaced volume of the storage containers. Exhaust emissions appear to be similar to those for propane (Lacy et al. 1984).

Some more or less experimental fleets are in operation but widespread use of CNG is a longer term option. However, the weight problem currently is no worse than that of electric vehicles, and if high energy density batteries can be developed in a reasonable period of time, electricity would unquestionably be the safer and cleaner option.

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APPENDIX 6

5/5

NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL

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Dr Keith Wilson
39 Bellevue Drive
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Dear Dr Wilson

As you requested by telephone, I am writing to pass on to you the outcome of the Public Health Committee's consideration of MMT at its meeting in Canberra on 3/4 September 1987.

In general, the decision was that there were no toxicological concerns over the use of MMT in petrol, but that the matter should be cleared through the Air Quality Committee in terms of vehicle emissions. This was to be expedited after the PHC meeting in order to resolve the matter as soon as possible.

Yours sincerely

DR G J MURPHY
SECRETARY
PUBLIC HEALTH COMMITTEE

15 October 1987

APPENDIX 7

ATTACHMENT 1

INSTITUTE FOR BASIC RESEARCH IN DEVELOPMENTAL DISABILITIES

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JUL 12 1990

July 6, 1990

Donald R. Lynam, Ph.D., CIH, PE
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Dear Don:

Thank you for the transcript of the hearing on the manganese (Mn) additive and the copies of comments and studies submitted by various persons regarding potential health affects of exposures to Mn. Based on what I heard at the public hearing and my review of these materials, I would like to make the following comments.

First, as you know, the study of miners exposed to chronic manganese overload revealed that many showed extrapyramidal symptoms including bradykinesia, postural difficulties, prominent rigidity, tremor and, occasionally, significant dystonia. In addition, psychiatric symptoms, referred to as "locura manganica" or manganese madness, were also typically observed. Taken together these data indicate that, in high doses, Mn is a neurotoxic atom. There is no evidence that these effects could occur at levels in the ambient atmosphere. To the best of my knowledge there is no evidence, and none has been cited, that established that children and pregnant women are more susceptible to manganese than males and non-pregnant females.

A questions was also raised regarding the impact of Mn on neurotransmitters. The type of changes in neurotransmitters involved in movement disorders are not clearly understood. Experimental data from various animal species indicate that Mn affects the neurotransmitters that, in humans, are disturbed in Parkinson's disease. For obvious reasons, such studies cannot be done on humans. However, signs and symptoms observed in miners exposed to Mn at high levels indicate that these neurotransmitters are affected in Mn-induced movement disorders. To my knowledge, there is no evidence to suggest that similar effects take place at lower Mn exposure levels.

I also concur with A. Barbeau's opinion (Neurotoxicology 5(1): 13-36, 1984) that chronic manganese intoxication is not the cause of a form of idiopathic Parkinson's disease. However, Mn can cause a PD-like syndrome, as shown in those studies examining chronic Mn overload.

Regarding the effect of Mn on the immune system, experimental data seem to support the hypothesis that Mn may affect the immune system, at higher levels. These data, however, are not conclusive. In humans, there are no good data to support this hypothesis. And there is no evidence that exposure to environmental levels of Mn (approximately 0.04 ug/m^3) affects human negatively. It is well documented that presently Mn exposure from food and water (3,000 ug/day) is much bigger than from air (0.04 ug/m^3).

During the EPA hearing, Ms. E. Silbergeld's main line of attack was: Ethyl added lead to gasoline in 1925 and this proved to be a powerful neurotoxic atom; today, Ethyl wants to add another metal. Today, in contrast to 1925, we know quite a lot about trace metals' effect on human health. In addition, we also have the EPA, EDF, and other state and city Agencies to monitor the environment. Based on Canadian experience and extensive research (e.g., the Health Effects Institute report on "Potential Health Effects of Manganese in Emissions from Trap-Equipped Diesel Vehicles"), there is no indication that additional manganese from the use of fuel additives will create health problems. According to the HEI report "under the worse-case assumptions, the contribution of manganese from automobile exhaust is not anticipated to be greater than 2.5 percent of the dietary intake. This small contribution of manganese from mobile sources is not expected to tax the homeostatic mechanisms that regulate the levels of manganese throughout the body's tissues." The contribution of Mn from use of the additive would be much less than that examined in the HEI report.

Dr. Donaldson's data on the mechanism of action of Mn are quite interesting but, in my opinion, not relevant to Ethyl's application. The statements by Mr. Hodges and some of those by Dr. Donaldson concentrated primarily on high risk or selective susceptibility cases. Because Mn is an ubiquitous atom and its daily intake is high, to the extent that the people have difficulty in handling Mn, they will develop problems irrespective of Ethyl adding Mn to its products. It will be particularly true if some of the conditions are genetically determined.

In summary, in my judgment, Ethyl provided enough evidence to show that adding Mn to their products will not negatively affect human life and the environment.
While further research is always possible with respect to extremely low level exposures to substances such as Mn, and indeed several interesting avenues for research have been

suggested, this should not stop us from introducing Mn to gasoline because the balance for and against doing so is clearly in favor of approving Ethyl's application.

Best regards.

Sincerely yours,

Henry M. Wisniewski, M.D., Ph.D.
Director

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Education: M.D., Medical School, Gdansk (Danzig), Poland, 1955; Ph.D., Medical School, Warsaw, Poland, Experimental Pathology, 1960; Docent, Neuropathology, Medical School, Warsaw, Poland, 1965.

Honors:
Weil Award, American Association of Neuropathologists, 1969
Moore Award, American Association of Neuropathologists, 1972
Career Scientist, Health Research Council of the City of New York
Association for the Help of Retarded Children, New York City (AHRC) Chapter award, 1984
President, American Assoc. of Neuropathologists, 1984
Welfare League, Letchworth Village Chapter, AHRC, award 1985
Benevolent Society for Retarded Children, Staten Island Chapter, 1986
Fellow, American Association for the Advancement of Science, 1989

Experience:
Director, NYS Institute for Basic Research in Developmental Disabilities, 1976--present;
Professor of Pathology (Neuropathology), SUNY Health Science Center at Brooklyn, 1976--present;
Director, MRC Demyelinating Diseases Unit, Newcastle upon Tyne, England, 1974-1976;
Research Assoc., Assistant, Associate and full Professor of Pathology (Neuropathology), Albert Einstein College of Medicine, 1966-1975;
Visiting Scientist, Laboratory of Neuropathology NINCDS, NIH (Dr. Klatzo), 1962-63;
Visiting Neuropathologist, Division of Neuropathology, University of Toronto, Canada (Dr. J. Olszewski) 1961-1962;
Assistant-Associate Professor, Head of Laboratory of Experimental Neuropathology, Associate Director of Institute of Neuropathology, Polish Academy

of Science, Warsaw, Poland, 1958-1966.

Publications: Author or co-author of 470 publications.

Societies:
American Association for the Advancement of Science
American Association of Neuropathologists
British Immunological Association
British Society of Neuropathology
Canadian Association of Neuropathologists
American Association for Retarded Citizens
Association for Research in Nervous and Mental Disease
American Association on Mental Deficiency
International Society for Developmental Neurosciences
Society for Experimental Neuropathology
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Major Research Interests: Developmental disabilities and dementia; Neuronal fibrous protein pathology; Demyelinating diseases.

Other Activities:
Past Member Neurology B Study Section, NIH. Ad hoc reviewer
Past Member National Institute of Child Health and Human Development (NICHD), NIH, Mental Retardation Research Committee, Ad hoc reviewer
Member of the Board of Directors of the Research Foundation for Mental Hygiene, Inc.
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Acta Neuropathologica (Berl.)
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Member of the Journal of Neural Transmission - Dementia Section
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Member of the Editorial Board of Brain Pathology

PUBLICATIONS

HENRYK M. WISNIEWSKI

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ATTACHMENT 2

MANGANESE : COMMENTS ON HUMAN HEALTH RISK

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For the general population, food usually constitutes the major source of manganese intake. The daily intake from food may vary over a wide range. In 1982, we have performed a duplicate meal study in Belgium and found values ranging from 0.6 to 8.8 mg manganese in 24-hr diets (median : 2.6). Drinking water generally contains less than 100 µg manganese/l (median value around 5 µg/l) but some mineralized water may contain higher concentrations. The median intake via drinking water is about 8 µg/day but can be as high as 2 mg/day from some water supplies. In many countries legislation requires that drinking water does not contain more than 50 µg manganese per liter. The adequate daily oral intake of manganese has been estimated at 2-3 mg but an additional amount of manganese (usually 1 mg po daily) is frequently prescribed during pregnancy in association with other oligoelements and various vitamins. Manganese absorption from the gastrointestinal tract is controlled by homeostatic mechanisms and varies from 1 to 5% (3% on the average). Individuals ingesting 3 mg manganese with food and drinking water will thus absorb on the average 90 µg manganese daily (range : 30 to 150 µg).